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Commentary

Cellular Mechanisms of Aging and Emerging Biotherapeutic Strategies

Marcus Langford*

Department of Molecular Gerontology, Zenith University of Life Sciences, Oslo, Norway

DESCRIPTION

This article explores the molecular and cellular mechanisms underlying aging, including genomic instability, mitochondrial dysfunction, protein misfolding, cellular senescence and stem cell exhaustion. It also discusses emerging biotherapeutic approaches such as senolytics, gene therapy and stem cell therapies that aim to mitigate these age-related processes and improve healthspan, Aging is characterized by the progressive decline of cellular, tissue and organ function, leading to increased susceptibility to diseases such as cardiovascular disorders, neurodegeneration and cancer. While aging has long been considered inevitable, contemporary research reveals that its biological mechanisms are modifiable. Key processes such as DNA damage, mitochondrial dysfunction and cellular senescence drive tissue deterioration.

This article provides a comprehensive overview of the molecular and cellular mechanisms that drive aging, including genomic instability, telomere shortening, mitochondrial dysfunction, protein misfolding, cellular senescence and stem cell exhaustion. It delves into how these processes collectively impair tissue function, weaken physiological resilience and increase susceptibility to age-related diseases such as cardiovascular disorders, neurodegeneration, diabetes and cancer. Beyond describing the mechanisms, the article highlights cutting-edge biotherapeutic interventions aimed at mitigating these effects. These include senolytic therapies to remove senescent cells, gene therapies to repair DNA damage and maintain telomere length, mitochondrial-targeted compounds to improve energy metabolism and stem cell-based regenerative approaches. By integrating the latest research in molecular biology and translational medicine, the article emphasizes the potential to slow, delay, or even partially reverse biological aging through targeted interventions.

Genomic instability and DNA damage

DNA is constantly subjected to damage from oxidative stress, radiation and replication errors. Over time, repair mechanisms become less efficient, allowing mutations to accumulate. Telomere shortening further limits regenerative potential. Gene-editing and gene therapy approaches are being explored to repair damaged DNA, maintain telomere length and restore cellular function.

Mitochondrial dysfunction

Mitochondria are critical for energy production, but aging reduces mitochondrial efficiency, leading to increased Reactive Oxygen Species (ROS). ROS accumulation damages DNA, proteins and lipids, contributing to systemic decline. Biotherapeutics such as mitochondrial-targeted antioxidants and NAD^+ precursors have shown potential in preclinical models to improve mitochondrial function and metabolic health.

Protein misfolding and proteostasis

Aging disrupts protein homeostasis, causing accumulation of misfolded or aggregated proteins. Autophagy and proteasome activity decline, exacerbating cellular stress. Drugs enhancing autophagy, small molecule chaperones and proteostasis regulators are being investigated to restore cellular protein quality and prevent neurodegenerative conditions.

Cellular senescence and senolytics

Senescent cells accumulate with age, secreting pro-inflammatory cytokines known as the Senescence Associated Secretory Phenotype (SASP). Chronic inflammation contributes to tissue dysfunction and disease. Senolytic therapies, which selectively eliminate senescent cells, have improved physical function, cardiovascular health and lifespan in animal studies. Human trials are underway.

Stem cell exhaustion and regenerative therapies

Stem cells lose proliferative capacity with age, impairing tissue regeneration. Stem cell transplantation, pharmacological activation of endogenous stem cells and tissue engineering aim to restore regenerative potential. Biotherapeutics targeting stem cell pathways have shown promise in preclinical and early clinical studies for rejuvenating aged tissues.

Correspondence to: Marcus Langford, Department of Molecular Gerontology, Zenith University of Life Sciences, Oslo, Norway, E-mail: m.langford@zenithuniv.no

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CONCLUSION

Aging results from interconnected molecular and cellular mechanisms including DNA damage, mitochondrial dysfunction, protein misfolding, cellular senescence and stem cell exhaustion. Biotherapeutic interventions such as senolytics, gene therapy and stem cell-based approaches offer significant potential to slow or reverse these processes. By targeting the biological roots of aging, these therapies could extend healthspan, enhance physical function and improve quality of life in older adults. Aging is a multifaceted process driven by interconnected molecular and cellular mechanisms, including DNA damage, telomere attrition, mitochondrial dysfunction, protein misfolding, cellular senescence and stem cell exhaustion. These hallmarks collectively impair tissue homeostasis, reduce regenerative capacity and increase the risk of chronic diseases.